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Cardiovascular and cortisol reactions to acute psychological stress under conditions of high versus low social evaluative threat: Associations with the Type D personality construct

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ABSTRACT

Objective: Social evaluative threat is an important factor in the cardiovascular response to mental stress. This study examined whether Type D personality, characterized by social inhibition and negative affectivity, is associated with an adverse cardiovascular response to social evaluative threat, thereby contributing to increased risk of cardiovascular disease. We compared physiological stress reactions of Type D and non-Type D individuals in settings varying in social evaluation characteristics.

Methods: 2300 students were screened for Type D personality, and 130 selected for a non-social stress exposure condition (31 Type D, 30 non-Type D: 52% female) or a condition high in social evaluative threat (35 Type D, 34 non-Type D: 55% female). Systolic (SBP) and diastolic blood pressure (DBP), heart rate (HR) and salivary cortisol were measured at rest and in response to stress.

Results: Social evaluative threat resulted in higher cardiovascular responses than the non-social challenge (SBP, p =.001, η^2 = .092; DBP, p = .006, η^2 = .058; HR, p = .006, η^2 = .059). The greatest cardiovascular stress reactions were exhibited by Type D participants in the high social evaluation condition; reflected in significant group x condition interactions for SBP, $F(1,126)$ =7.29, p =.008, η^2 =.055, DBP, $F(1,126)$ =5.23, p =.024, η^2 =.040, and HR, $F(1,126)$ =5.04, p =.027, η^2 =.038, reactivity. Only Type Ds in the social condition mounted a positive cortisol response, $F(1,33)$ =5.07, p =.031, η^2 =.133.

Conclusions: It would appear Type D individuals show different stress reactions depending upon the social evaluative nature of the stress exposure, with this dysregulation of the stress response potentially increasing cardiovascular disease risk.

Keywords: Type D personality; social evaluation; cardiovascular reactivity; cortisol reactivity; psychological stress.

SBP- systolic blood pressure; **DBP**- diastolic blood pressure; **HR**- heart rate; **NA**- negative affectivity; **SI**- social inhibition; **SAM**- sympathetic-adrenal-medullary system; **HPA**-hypothalamic-pituitary-adrenocortical axis; **BMI**- body mass index; **DS14**- Type D scale-14; **PASAT**- paced auditory serial addition test.

INTRODUCTION

Exaggerated biological reactions to stress have generally been viewed as maladaptive with some evidence to show that that individuals with exaggerated cardiovascular stress responses are at increased risk of developing cardiovascular disease due to various manifestations such as hypertension (1, 2), systemic atherosclerosis (3), left ventricular hypertrophy (4, 5), coronary artery calcification (6), as well as increased cardiovascular disease mortality (7). Additionally, exaggerated cortisol reactivity has been associated with coronary artery calcification (8) and increased hypertension (9) and cardiovascular disease risk (10). Moreover, social threat is considered to be a major component in stress-induced reactivity with evidence to show that social evaluation *per se* increases cardiovascular responses to stress (11-13). A similar pattern emerges for cortisol reactivity(14). Further it has been demonstrated that social-evaluative threat is required to elicit activation of the HPA-axis (15-17). Indeed, a dose-dependent increase in cardiovascular and cortisol reactivity to a speech stress task was observed under conditions of increasing social-evaluative threat, as a consequence of increasing audience size during a speech stressor (18). Thus, the overall consensus suggests that increasing social-evaluation perturbs increased cardiovascular and cortisol stress reactivity.

Type D, ‘distressed’, personality is characterised by the tendency to inhibit emotions in social situations (social inhibition: SI) and the propensity to experience high levels of negative emotion (negative affectivity: NA) (19) and has been implicated in the development of cardiovascular disease, although the evidence is now somewhat mixed (20). Extant findings indicate an association between Type D and increased risk of mortality in existing coronary artery disease patients (20, 21), as well as increased risk of developing coronary heart disease in healthy populations (22). However, a recent meta-analysis suggests that earlier Type D studies

may have overestimated its prognostic significance and there may not always be significant effects of the SI x NA interaction when controlling for first order effects (20). In addition, the underlying mechanisms of any putative association between Type D and cardiovascular disease remain unclear, although dysregulation of the sympathetic-adrenal-medullary (SAM) system and hypothalamic-pituitary-adrenocortical (HPA) axis, as evidenced by exaggerated physiological reactions to acute stress, have been proposed.

Previous studies investigating the cardiovascular and cortisol response to acute psychological stress in Type D individuals have yielded inconsistent findings. In response to a mental arithmetic challenge, exaggerated SBP reactivity was associated with increased SI, whereas blunted HR responses were related to, albeit non-significantly, high NA in undergraduate males (23). Additionally, both high SI and NA were associated with exaggerated cortisol reactivity (23). Further, in comparison to non-Type D college students, males with Type D personality exhibited exaggerated cardiac output responses during a mental arithmetic task, with no differences in blood pressure or HR reactivity (24). Conversely, lower HR and cardiac output reactivity has been reported in Type D females, again with no differences in SBP or DBP responses (25).

A potential reason for the mixed findings for Type D may be the social nature of the stress tasks. This is pertinent because the SI component of Type D refers to the inhibition of emotions in social situations. Emotional inhibition has been shown to relate to exaggerated cardiovascular (26) and cortisol (27) stress reactions. For example, using a serial mathematical subtraction task designed to be socially evaluative with elements of harassment, reward, and overt performance monitoring, individuals with high SI showed exaggerated blood pressure and cortisol reactivity (23). On the other hand, no differences between Type D and non-Type D

participants in blood pressure or HR stress responses have been observed when a mental arithmetic stressor but with minimal social evaluation was employed (24). Further, diminished HR and cardiac output reactivity were observed in Type D individuals during a serial subtraction task without social elements that used a non-verbal keypad response and the experimenter scored performance behind an opaque screen (25). Taken together, these studies support the contention that Type D individuals may exhibit exaggerated cardiovascular and cortisol reactions to socially evaluative stressors, but not in non-social conditions.

Given the potential role of the social nature of the stressor used in explaining the previous mixed associations between Type D personality and physiological stress reactivity, the current study, was designed to compare cardiovascular and cortisol stress reactions of Type D individuals with individuals who were non-Type D in two settings varying in social evaluation characteristics. It was expected that increased social evaluation would enhance cardiovascular and cortisol reactivity regardless of Type D classification, and that the enhanced stress reactivity to socially evaluative stress would be particularly evident among individuals with Type D personality.

METHODS

Participant characteristics

A questionnaire screening for Type D personality was administered to 2300 University of Birmingham students (1350 women) via e-mail and online recruitment. Based on screening scores, 130 (66 Type D) healthy participants were invited to attend a laboratory session which consisted of either a social or non-social stress testing condition. The mean (SD) age of the

selected sample was 20.5 (1.87) years and their mean (SD) body mass index (BMI) was 22.8 (2.94) kg/m². Table 1 presents the socio-demographics and health behaviours of the four sub-groups (Type D social, Type D non-social, non-Type D social, non-Type D non-social). The majority of the participants indicated they were "white" (89%). None had a history of cardiovascular disease, a current illness or infection, or were taking medication, with the exception of three individuals using anti-depressive medication: two Type D non-social and 1 Type D social. All participants provided written informed consent and the study was approved by the University of Birmingham ethics committee. Data collection took place between November 2012 and March 2013.

Procedure

To ensure accurate Type D classification, the current study used more stringent cut-off criteria than those suggested by Denollet (1): a DS14 score of ≥ 14 and ≤ 8 on both the SI and NA subscales classified Type D and non-Type D, respectively. These cut-offs were based upon the upper and lower quartiles of our sample, as although median splits (23, 25, 28) have also been employed to create Type D dichotomies, this can create a risk of misclassification and is generally advised against (29). Type D and non- Type D individuals were randomly allocated to either the social or non-social condition. The study employed a single-blind testing procedure with regard to Type D status. Laboratory sessions commenced at 13:30, 15:30 or 17:30. Prior to testing, participants were requested to refrain from eating for 1hr, drinking caffeine or smoking for 2hr, and from physical exercise and drinking alcohol for 12hr. Participants' height and weight were measured, and BMI subsequently calculated, and they completed the questionnaire pack, including a further DS14 to ensure correct Type D classification and test-retest reliability. During the adaptation period, participants lay in a semi-recumbent position, and remained in that

position throughout the session. A blood pressure cuff was attached and participants then reclined quietly for 10- minutes. This was followed by a formal 10-minute resting baseline before the stress tasks. Following each task, participants provided ratings of subjective impact and social evaluation. During the 5-minute inter-task period participants lay quietly. To exclude any social evaluation from the experimenter initiating physiological measures, participants were informed that the experimenter would be in attendance only to undertake physiological assessment and was not concerned with task performance.

Acute psychological stress task

The 5-minute modified Stroop colour-word interference task was presented on a computer screen and required participants to identify the incongruent colour in which a target word was presented by selecting, one of four identifier words naming the colour (30). Performance was titrated to ~50% to control for individual differences in task performance and percentage of correct responses was recorded as a check. Responses were executed on a keypad. An error resulted in an 'X', and a response exceeding ~5-second time limit resulted in a 'Too late' message on screen, both outcomes being accompanied by a short auditory beep. The number of trails could as to keep performance titrated to around 50% those who provided more correct responses were given a 'Too late' message more quickly thus although the time period was set to 5 minutes, the actual number of trails could vary. The Stroop was followed by a 5-minute inter-task rest period and then the 10-minute Paced Auditory Serial Addition Test (PASAT) (31), which has been shown to perturb both cardiovascular and salivary cortisol activity (32, 33), and demonstrates good test-retest reliability (34). Participants are presented, via a CD player, with a series of single digit numbers and are required to add the present number to the previously presented number, and report their answer aloud. They then have to remember the

last number they heard in order to add it to the next number read out from the CD. During the last five numbers of each block of 10 numbers, a brief burst of loud aversive noise was presented; all participants received 21 noise bursts. Instructions for both tasks were presented via a video on a computer screen followed by a short practice. To maintain engagement for both tasks participants were informed they would start with 1000 points with 5 points deducted for each wrong answer.

Social manipulation conditions

Non-social condition: To maintain consistency in the auditory beeps received but to minimise social evaluation, during the Stroop and PASAT tasks respectively, the beeps were relayed via the computer programme and CD player. To maintain engagement for both tasks, participants, although told that they were not being evaluated, were urged to perform to the best of their ability. The only individual present was the experimenter measuring physiological activity. Performance score on the PASAT was calculated from a Dictaphone recording which the participant was unaware of.

Social condition: To introduce social evaluation and comparison for both tasks, participants were told that their performance was being assessed and they were in direct competition with fellow participants. They were also informed that their scores would be displayed on a prominent leader board, which they could see, and that they should attempt to beat the scores currently displayed. Participants were also filmed and this was displayed live on a television screen, which they were requested to remain focused on during the PASAT. They were also told that the recording would be assessed by “body language experts”, although no such assessment was undertaken. Following task instruction and practice, an additional

experimenter wearing a laboratory coat entered the room and stood in close proximity to participant to observe them obtrusively. The additional experimenter left the room following each task. During the Stroop test, the experimenter sounded a buzzer each time they gave an incorrect answer or exceeded 5 seconds before responding. During the PASAT, the experimenter conspicuously scored the participants and sounded a buzzer once during the last five numbers of each block of 10 numbers, mostly corresponding with an error or hesitation. The amount of auditory beeps presented in the social and non-social conditions were exactly the same; only the method of delivery varied.

Cardiovascular and salivary cortisol measures

The laboratory session consisted of six periods; 10-minute adaptation, 10-minute baseline, 5-minute Stroop Task, 5-minute inter-task period, 10-minute PASAT stress task, and 10-minute recovery. SBP, DBP and HR were measured discontinuously using a semi-automatic sphygmomanometer (Omron, IL) at minute 1, 3, 5, 7 and 9 during baseline and PASAT, and at minute 1 and 3 during the Stroop task. A single measure was also taken during the adaptation period for familiarity, although this measure was discarded. Two stimulated 2-minute saliva samples were obtained using salivettes at minute 8 of baseline and 8-minutes into the recovery period. Salivettes were centrifuged for five minutes at 4000rpm before being stored at -20°C until assay. ELISA kits (IBL International, Germany) were used to analyse all cortisol samples in duplicate. The mean intra-assay coefficient of variation was 9.9% and the inter-assay coefficient was 4.5%. Due to collection difficulties with one Type D participant in the social condition, cortisol assays were analysed for 129 participants.

Questionnaires

Type D personality

Type D personality was assessed using the Type D Scale-14 (DS14) (19) which comprises two 7-item subscales, measuring SI, e.g., “I find it hard to start a conversation”; “I am a closed kind of person” and NA, e.g., “I am often in a bad mood; “I often make a fuss about unimportant things”. Respondents indicate their answers on a 5-point Likert scale ranging from 0, false, to 4, true. These subscales are summed to yield an overall measure of Type D. A score of ≥10 on both SI and NA subscales has been used to indicate Type D personality classification (19, 35). Both the SI and NA subscales have shown good test-retest stability, r = .82 and .72 respectively, and high internal validity, Cronbach’s alpha = 0.86 and 0.88, respectively (19). The present study found a Cronbach’s α of .94 and .95 for the SI and NA scales respectively.

Health behaviours and depression

A questionnaire adapted from the Whitehall II study (36) was administered to measure average daily smoking (0, 1-5, 6-10, 11-20, 21-40, 41+ cigarettes per day) and weekly alcohol intake (0, 1-5, 6-10, 11-20, 21-40, 41+ units per week) which were subsequently dichotomised to current/no smoker and <11/≥11 weekly units. In order to calculate cardio-respiratory fitness, participants indicated how much time they spent in activities of different intensities which were allocated category scores from 1 to 5, where 1 indicates inactivity and 5 indicates participation in brisk exercise for over 3 h per week, with the physical activity levels of 1, 2, 3, 4, and 5 assigned scores of .00, .32, 1.06, 1.76, and 3.03, respectively (37). The following formula was used to calculate cardio-respiratory fitness in METS: (0 if female or 2.77 if male) − ((age × 0.10) − ((BMI) × 0.17) − ((resting heart rate) × 0.03) + (physical activity score) +18.07 (37).

The seven item depression subscale of the Hospital Anxiety and Depression Scale (HADS) (38) was used to measure depressive symptoms. Responses are indicated on a 4-point scale, ranging 0-3, with higher scores indicating greater depression. Psychometric analysis indicates good test-retest reliability with a coefficient of .85 (39), and Cronbach's alpha's of .90 (40).

Psychological stress task questionnaire

Participants indicated perceived stressfulness and task engagement immediately following each stress task by using a 7 point Likert-type scale ranging 0, not at all to 6, extremely. These scales have been used successfully in previous studies investigating physiological reactivity to acute psychological stress (32, 41). To check the success of the social evaluation manipulation, following each stress task, participants indicated on a 7 point Likert-type scale, ranging 0, not at all to 6, extremely, the extent to which they felt they were being socially evaluated by others.

Data Analysis

For the cardiovascular measures, averages of the baseline and stress (combined average of Stroop and PASAT) periods were calculated. Reactivity scores were calculated by subtracting baseline from stress averages. Repeated measures ANOVAs were performed to confirm the stress tasks perturbed cardiovascular and cortisol activity. Group differences in socio-demographics, health behaviours, depression, stress task perceptions and social manipulation ratings (both averaged across tasks), performance, number of trails, and baseline cardiovascular and cortisol variables were tested using 2 (Type D versus non-Type D group) x 2 (social versus non-social condition) ANOVAs for continuous variables, and chi-square for categorical

variables. Similar 2 x 2 ANOVAs were conducted to analyse group differences in physiological reactivity. 2 x 2 ANCOVAs were utilised to determine whether group differences withstood adjustment for potential confounding variables. Pairwise comparisons were undertaken to elucidate significant differences. Partial η^2 is reported as an index of effect size throughout. *p*-values of $\leq .05$ were considered statistically significant.

RESULTS

Participant characteristics

All participants allocated to the Type D and non-Type D groups conformed to the respective cut off criteria of ≥ 14 and ≤ 8 on the SI and NA subscales. Consistent with the allocation procedure, the SI and NA subscale scores, and the Total DS14 scores, were higher for Type D than for non-Type D participants (Table 1). There were no differences in the SI and NA scores when comparing the social threat vs. non-social threat conditions. This pattern of results was identical for the scores on the DS14 completed in the laboratory to confirm the robustness of group allocation. The test-retest scores over an average 4-month period were r = .92 and .93, for the SI and NA scales, respectively.

The summary data for socio-demographics, health behaviours and depression are presented in Table 1. Group differences emerged only for age, estimated cardio-respiratory fitness and depression, $F(1,126) = 8.10$, $p = .005$, $\eta^2 = .060$; $F(1,126) = 11.56$, $p = .001$, $\eta^2 = .084$; $F(1,124) = 70.26$, $p < .001$, $\eta^2 = .362$, respectively; Type D individuals were slightly older, had lower cardio-respiratory fitness, and scored higher on the depression subscale. The main effects for

condition and group x condition interactions for these variables were not statistically significant ($p > .060$).

Social manipulation

As expected, social evaluation ratings differed between conditions, $F(1,125) = 46.30$, $p < .001$, $\eta^2 = .270$. Participants in the non-social condition ($M\pm$S.D: Type D = 3.3$\pm$1.50. Non-Type D = 2.3$\pm$1.57) rated the tasks as low-to-moderate in terms of social evaluation, whereas participants in the social condition ($M\pm$S.D: Type D = 4.7$\pm$1.16. Non-Type D = 4.4$\pm$1.47) rated the tasks as moderate-to-highly socially evaluative. There was also a significant main effect of Type D group, $F(1,125) = 7.69$, $p = .006$, $\eta^2 = .058$, with Type D individuals overall reporting the tasks, irrespective of condition, as more socially evaluative. There was no group x condition interaction effect ($p = .15$).

Stress task ratings and performance

Summary stress task ratings and performance data are also presented in Table 1. There were no significant main effects or interactions for self-reported engagement or PASAT total score indicating no overall differences in task engagement ($p > .050$). Additionally, both the mean number of Stroop trails presented (M= 106.9%, S.D= 12.53) and the correct mean Stroop response rate (M= 57.5% , S.D= 6.35) showed no main effects or interactions indicating successful titration and no group differences in the task. Although there was no condition effect or group x condition interaction ($p > .19$ in both cases) for rated stressfulness, Type D participants perceived the tasks as more stressful than their non-Type D counterparts, $F(1,125) = 8.18$, $p = .005$, $\eta^2 = .061$.

[Insert Table 1 about here]

Cardiovascular stress reactions

Table 2 presents the summary data: there were no group differences in any of the baseline cardiovascular values ($p > .16$). Repeated measures ANOVAs confirmed that the stress tasks perturbed all cardiovascular parameters: SBP, $F(1,129) = 293.95.70$, $p < .001$, $\eta^2 = .695$; DBP, $F(1,129) = 489.23$, $p < .001$, $\eta^2 = .791$; HR, $F(1,129) = 262.04$, $p < .001$, $\eta^2 = .671$. There were no group main effects for any of the cardiovascular reactivity variables ($p > .30$ in all cases). However, the social condition proved more provocative than the non-social condition: for SBP, $F(1,126) = 12.70$, $p = .001$, $\eta^2 = .092$; for DBP, $F(1,126) = 7.72$, $p = .006$, $\eta^2 = .058$; for HR, $F(1,126) = 7.88$, $p = .006$, $\eta^2 = .059$. Importantly, the group x condition interaction was significant in each case: for SBP, $F(1,126) = 7.29$, $p = .008$, $\eta^2 = .055$; for DBP, $F(1,126) = 5.23$, $p = .024$, $\eta^2 = .040$; for HR, $F(1,126) = 5.04$, $p = .027$, $\eta^2 = .038$. These interaction effects are illustrated in Figure 1. For both HR and blood pressure, the Type D participants in the social condition had significantly higher reactivity than the non-social condition Type D participants ($p \leq .050$ in all cases). In addition SBP and HR reactivity was greater for the Type D participants in the social condition than the non-Type D participants in both the social and non-social conditions ($p < .035$ in all cases). Within the non-social condition, Type D individuals displayed slightly lower DBP reactivity ($p = .054$) than non-Type D participants; for the same comparison there were similar indications of attenuated SBP reactivity for Type Ds in the non-social condition ($p = .091$). Similarly, within the non-social condition, compared to their non-Type D counterparts, Type D participants had lower HR reactivity, although this was not significant ($p = .41$). Finally, both SBP and DBP reactivity ($p < .020$ in both cases) for Type D individuals in the non-social condition was significantly attenuated relative to that shown by non-Type D participants in the social condition.

[Insert Table 2 and Figure 1 about here]

Cortisol stress reactions

Table 2 presents the summary data: baseline cortisol levels did not differ between groups (p = .14), and overall the stress tasks failed to increase cortisol concentration (p = .99). There were, however, significant main effects for both group, $F(1,125) = 7.87$, p = .006, η^2 = .059, and condition, $F(1,125) = 5.31$, p = .023, η^2 = .041, for cortisol change; Type D participants were more reactive and the social condition was more provocative. The group x condition interaction effect did not meet the criterion for statistical significance (p = .16). The summary cortisol data are presented in Figure 2. As can be seen, there was a positive cortisol reaction only for Type D participants in the social condition; pairwise comparisons indicated that the cortisol change score for Type D individuals in the social condition differed from that observed for the other three groups ($p < .050$ in each case). As a sensitivity analysis, repeated measures ANOVAs comparing baseline and stress cortisol concentrations were conducted separately for each of the four groups. The only group to demonstrate a significant increase in cortisol was the Type D participants in the social condition, $F(1,33) = 5.07$, p = .031, η^2 = .133; the non-Type D non-social participants actually showed a decrease, $F(1,29) = 8.42$, p = .007, η^2 = .225. For Type D non-social (p = .50) and non-Type D social participants (p = .25) cortisol did not change significantly from baseline to stress exposure.

[Insert Figure 2 about here]

Covariate analyses

As there were sub-group differences in age, cardio-respiratory fitness, depression and perceived stressfulness, the main analyses were repeated, adjusting for these variables in addition

to the appropriate baseline cardiovascular and cortisol levels. The SBP, $F(1,118) = 5.45$, $p = .021$, $\eta^2 = .044$, DBP, $F(1,118) = 4.01$, $p = .048$, $\eta^2 = .033$, and HR, $F(1,118) = 4.27$, $p = .041$, $\eta^2 = .035$, reactivity group x condition interactions withstood covariate adjustment. Similarly, for cortisol reactivity the main effect of condition, $F(1,117) = 6.58$, $p = .012$, $\eta^2 = .053$ was preserved, although the main effect of group was reduced to a trend, $F(1,117) = 3.04$, $p = .084$, $\eta^2 = .025$. Additionally controlling for anti-depressant medication use did not alter these outcomes. Similarly, although there were no group differences in BMI ($p = .78$), sex ($p = .74$), or smoking status ($p = .41$), controlling for these potential confounders did not change the outcomes. There were no differences in any of the physiological reactivity variables across the three testing times (all p's > .055) and controlling for testing time did not change any of the outcomes.

DISCUSSION

The present study was the first we are aware of to compare cardiovascular and cortisol stress reactions of extreme Type D and non-Type D individuals in two settings varying in social evaluation characteristics. As expected, the condition high in social evaluative threat elicited greater cardiovascular and cortisol reactions, irrespective of the Type D status of the participants; this very much resonates with the findings of previous research into the physiological impact of social evaluation (11-13, 18, 42). Importantly, given the primary aim of the current study, this enhanced reactivity was a particular feature of Type D participants. Whereas non-Type D individuals in the social and non-social conditions exhibited cardiovascular reactions of a comparable magnitude, Type D individuals in the social condition were characterised by greater

SBP, DBP and HR reactions than their non-social Type D counterparts. Regarding cortisol stress reactions, only Type D individuals in the social condition mounted a positive cortisol response. Overall, it would appear that relative to non-Type Ds, Type D individuals showed heightened reactivity to social-evaluative threat, but not in response to the non-social task. The pattern of results was still evident following statistical adjustment for potential confounders; age, cardio-respiratory fitness, depression, perceived stressfulness and baseline physiological levels.

As indicated, previous studies of stress reactivity and Type D personality have produced mixed results. The findings of the present study would appear to go some way toward accounting for these discrepancies. Type D individuals seem to be characterised by higher cardiovascular and cortisol reactivity mainly when exposed to stress tasks high in social evaluative threat (23). Where the stress exposure is largely non-social, Type D individuals either do not differ from their non-Type D counterparts in terms of cardiovascular stress reactivity (24) or actually show diminished cardiovascular reactions (25). Our finding of Type D individuals within the non-social condition showing no difference to non-Type Ds stress reactivity is in line with this.

It should also be noted that Type D individuals, regardless of the social evaluative condition, reported the tasks to be more stressful. Indeed, research has suggested that Type D individuals may have a cognitive bias towards interpreting threat and this may increase their vulnerability to perceived social stress (43). This is supported by neuroimaging data which has shown the two components of Type D personality, SI and NA, are associated with unique brain activity patterns in response to perceived socially threatening stimuli (44). Interestingly, despite the Type D individuals in the non-social condition reporting the tasks as significantly more stressful, they had comparable SBP, DBP and HR reactions to their non-Type D counterparts. This is in line with previous personality research which has demonstrated that there is a paradox

between subjective self-report ratings and physiological responses (45). It would appear that there may be further thematic links between Type D emotional patterns and the particular physiological response profiles observed here, thus it would be recommended for future research to investigate this further.

Interest in Type D personality arises from its association with increased risk for cardiovascular disease morbidity (22) and mortality (20, 21). Dysregulation of the SAM system and HPA-axis has been implicated (46) and there is now evidence that those who show exaggerated cardiovascular stress responses are at increased risk of developing cardiovascular disease morbidity (1-6) and mortality (7). In addition, exaggerated cortisol reactivity has been associated with coronary artery calcification (8), hypertension (9) and cardiovascular disease risk (10). Accordingly, the increased stress reactions observed in Type D individuals when exposed to situations high in social evaluation may increase their risk of cardiovascular disease. This is particularly pertinent given that most of the stressors that people face in life are social rather than non-social (14, 47). Similarities between the increased stress reactivity in Type D individuals under social evaluative stress can also be made with the concept of interpersonal sensitivity; a stable trait characterized by ongoing concerns about negative social evaluation (48). Individuals high in interpersonal sensitivity are vigilant and sensitive to others' evaluation, and consequently adopt defensive behaviours such as SI to avoid negative social evaluation. Indeed, like Type D personality, a recent integrative review has demonstrated individuals with high interpersonal sensitivity may be at increased risk of cardiovascular disease, although unmeasured or poorly measured potential confounders suggest caution in attributing direct causality (48). Given that Type D and non-Type D individuals did not significantly differ in their responses during non-social stress, and provided some inclinations towards diminished reactivity, this may prove a

valuable area for future research. If Type D individuals display attenuated reactivity under non-social conditions it may suggest that it is the departure of their reactivity profile from the norm, i.e., their facility to exhibit extreme responses, potentially diminished when faced with non-social stress exposures and exaggerated when faced with stressors high in social evaluation. From another perspective, if Type D personality is considered to be a continuous variable, then it may actually be most adaptive around a mid-point, with high or low extremes representing the maladaptive tails of a normal distribution.

The present study is not without limitations. First, although the Type D personality construct has classically been treated as a dichotomised variable, based on a median split on the DS14 subscales (19), more recent research has suggested that Type D personality may be best viewed as continuous rather than categorical (20, 49, 50). For example, a meta-analysis reported that the use of a categorical approach may have over-estimated the prognostic value of Type D (20). However, it should be noted that recent research has reported that both the interaction of continuous SI and NA scores (continuous measure of Type D) and the Type D versus non-Type D classification defined by cut-off scores were both associated with an increased risk of cardiovascular events (51). Similarly, a study examining stress reactivity found that using either a continuous or categorical measure of Type D personality produced analogous associations with cardiovascular reactions (25). A categorical approach can also reduce power and increases the risk of false positives (52), and so caution may be necessary in interpreting the results. However, a strength of the current study was the use of more stringent classification criteria than previous studies (24, 28), with $\geq$14 and $\leq$8 classifying Type D and Non-Type D groups, respectively; this would serve to limit the possibility of misclassification. Additionally, test-retest scores over an average 4-month period were r = .92 and .93, for the SI and NA scales, respectively, indicating

strong stability. Regarding reduced power it should be noted that the current study included a larger sample size than previous studies of reactivity and Type D (24, 25, 28). Second, given that it has been stated that Type D personality is more than just NA, and that SI is an important moderator of the effects of negative emotions on adverse clinical outcomes (53), it would have been informative to analyse the separate effects of SI and NA, and the potentially important synergistic effects of the SI x NA interaction term on reactivity (23, 49, 53). Clearly the current design precludes this and the SI and NA subscales for the selected participants were highly correlated both at screening ($r = .92$, $p < .001$) and in the laboratory ($r = .85$, $p < .001$). Accordingly, determining the separate and interactive contributions of NA and SI to reactivity differences was not possible. Given the issues with dichotomising variables such as reduced power and misclassification, there has been a clear emphasis on moving away from this practice (49, 50) . Consequently, future research should implement a design where it would be possible to assess the conceptually important SI x NA interaction and the first order effects of SI and NA on stress reactivity. Third, the associations between Type D personality and adverse disease outcomes has received some criticism, citing failures to replicate earlier results and the likelihood of confounding (20, 50, 54). It should be noted, however, that the current study controlled for a number of potential confounders including depression. Fourth, it could be argued that the manipulations in the social stress condition, i.e. presence of evaluators, scoring, and live recording, may have also increased factors such as objective self-awareness and competition which may have contributed to the differential reactivity profiles. However, the participants in the social condition rated it as significantly more socially-evaluative ($p < .001$) and research has shown that social evaluative threat per se often includes elements of self-awareness and competition (16). Fifth, we employed a between-subject design whereby Type D and non-Type

D individuals were assigned to either a social or non-social condition. This was purposive as we wanted to avoid the risk of habituation of reactivity that may have arisen from subjecting the same participants to two stress exposures (28, 55). Further, participants' DS14 scores and demographics were matched according to condition assignment and, importantly and consequentially, there were no group x condition interactions for any of these variables.

In summary, the present analyses indicated that Type D individuals exhibit exaggerated cardiovascular and cortisol reactivity under conditions of high socially evaluative threat, but comparable cardiovascular responses in non-social stress conditions. These results help resolve some of the previous inconsistencies in the literature and suggest Type D individuals may exhibit exacerbated or comparable, potentially attenuated, stress reactions depending on the social evaluative nature of the stressor. Thus, dysregulation of the SAM system and HPA-axis with exaggerated responses during social may contribute to the association between Type D personality and increased cardiovascular disease risk. Future research should utilise a continuous Type D component given the fact that it is not possible to discern from the present study whether it is SI, NA, or the SI x NA interaction that is at the core of the Type D model as initially described by Denollet and colleagues (19), and accounts for the association with physiological responses observed here.

REFERENCES

1. Carroll D, Ginty AT, Painter RC, Roseboom TJ, Phillips AC, de Rooij SR. Systolic blood pressure reactions to acute stress are associated with future hypertension status in the Dutch Famine Birth Cohort Study. Int J Psychophysiol 2012;85:270-3.
2. Carroll D, Ring C, Hunt K, Ford G, Macintyre S. Blood pressure reactions to stress and the prediction of future blood pressure: effects of sex, age, and socioeconomic position. Psychosom Med 2003;65:1058-64.
3. Everson SA, Lynch JW, Chesney MA, Kaplan GA, Goldberg DE, Shade SB, Cohen RD, Salonen R, Salonen JT. Interaction of workplace demands and cardiovascular reactivity in progression of carotid atherosclerosis: population based study. BMJ 1997;314:553-8.
4. Kapuku GK, Treiber FA, Davis HC, Harshfield GA, Cook BB, Mensah GA. Hemodynamic function at rest, during acute stress, and in the field: predictors of cardiac structure and function 2 years later in youth. Hypertension 1999;34:1026-31.
5. Murdison KA, Treiber FA, Mensah G, Davis H, Thompson W, Strong WB. Prediction of left ventricular mass in youth with family histories of essential hypertension. Am J Med Sci 1998;315:118-23.
6. Matthews KA, Zhu S, Tucker DC, Whooley MA. Blood pressure reactivity to psychological stress and coronary calcification in the Coronary Artery Risk Development in Young Adults Study. Hypertension 2006;47:391-5.
7. Carroll D, Ginty AT, Der G, Hunt K, Benzeval M, Phillips AC. Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. Psychophysiology 2012;49:1444-8.

8. Hamer M, O'Donnell K, Lahiri A, Steptoe A. Salivary cortisol responses to mental stress are associated with coronary artery calcification in healthy men and women. Europ Heart J 2010;31:424-9.
9. Hamer M, Steptoe A. Cortisol responses to mental stress and incident hypertension in healthy men and women. J Clin Endocrinol Metab 2012;97:29-34.
10. Girod JP, Brotman DJ. Does altered glucocorticoid homeostasis increase cardiovascular risk? Cardiovasc Res 2004;64:217-26.
11. Allen KM, Blascovich J, Tomaka J, Kelsey RM. Presence of human friends and pet dogs as moderators of autonomic responses to stress in women. J Pers Soc Psychol 1991;61:582-9.
12. Smith TW, Nealey JB, Kircher JC, Limon JP. Social determinants of cardiovascular reactivity: effects of incentive to exert influence and evaluative threat. Psychophysiology 1997;34:65-73.
13. Wright RA, Tunstall AM, Williams BJ, Goodwin JS, Harmon-Jones E. Social evaluation and cardiovascular response: an active coping approach. J Pers Soc Psychol 1995;69:530-43.
14. Dickerson SS, Gruenewald TL, Kemeny ME. When the social self is threatened: shame, physiology, and health. J Pers 2004;72:1191-216.
15. Dickerson SS, Mycek PJ, Zaldivar F. Negative social evaluation, but not mere social presence, elicits cortisol responses to a laboratory stressor task. Health Psychol 2008;27:116-21.
16. Gruenewald TL, Kemeny ME, Aziz N, Fahey JL. Acute threat to the social self: shame, social self-esteem, and cortisol activity. Psychosom Med 2004;66:915-24.

17. Kirschbaum C, Pirke KM, Hellhammer DH. The 'Trier Social Stress Test'- a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology 1993;28:76-81.
18. Bosch JA, de Geus EJ, Carroll D, Goedhart AD, Anane LA, van Zanten JJ, Helmerhorst EJ, Edwards KM. A general enhancement of autonomic and cortisol responses during social evaluative threat. Psychosom Med 2009;71:877-85.
19. Denollet J. DS14: standard assessment of negative affectivity, social inhibition, and Type D personality. Psychosom Med 2005;67:89-97.
20. Grande G, Romppel M, Barth J. Association between type D personality and prognosis in patients with cardiovascular diseases: a systematic review and meta-analysis. Ann Behav Med 2012;43:299-310.
21. Denollet J, Schiffer AA, Spek V. A general propensity to psychological distress affects cardiovascular outcomes: evidence from research on the type D (distressed) personality profile. Circ Cardiovasc Qual Outcomes 2010;3:546-57.
22. Svansdottir E, Denollet J, Thorsson B, Gudnason T, Halldorsdottir S, Gudnason V, van den Broek KC, Karlsson HD. Association of type D personality with unhealthy lifestyle, and estimated risk of coronary events in the general Icelandic population. Eur J Prev Cardiol 2013;20:322-30.
23. Habra ME, Linden W, Anderson JC, Weinberg J. Type D personality is related to cardiovascular and neuroendocrine reactivity to acute stress. J Psychosom Res 2003;55:235-45.
24. Williams L, O'Carroll RE, O'Connor RC. Type D personality and cardiac output in response to stress. Psychol Health 2009;24:489-500.

25. Howard S, Hughes BM, James JE. Type D personality and hemodynamic reactivity to laboratory stress in women. Int J Psychophysiol 2011;80:96-102.
26. Gross JJ, Levenson RW. Hiding feelings: the acute effects of inhibiting negative and positive emotion. J Abnorm Psychol 1997;106:95-103.
27. Lam S, Dickerson SS, Zoccola PM, Zaldivar F. Emotion regulation and cortisol reactivity to a social-evaluative speech task. Psychoneuroendocrinology 2009;34:1355-62.
28. Howard S, Hughes BM. Type D personality is associated with a sensitized cardiovascular response to recurrent stress in men. Biol Psychol 2013;94:450-5.
29. Veiel HO. Base-rates, cut-points and interaction effects: the problem with dichotomized continuous variables. Psychol Med 1988;18:703-10.
30. Gianaros PJ, Sheu LK, Matthews KA, Jennings JR, Manuck SB, Hariri AR. Individual differences in stressor-evoked blood pressure reactivity vary with activation, volume, and functional connectivity of the amygdala. J Neurosci 2008;28:990-9.
31. Gronwall DM. Paced auditory serial-addition task: a measure of recovery from concussion. Percept Mot Skills 1977;44:367-73.
32. Ginty AT, Phillips AC, Higgs S, Heaney JL, Carroll D. Disordered eating behaviour is associated with blunted cortisol and cardiovascular reactions to acute psychological stress. Psychoneuroendocrinology 2012;37:715-24.
33. Ring C, Burns VE, Carroll D. Shifting hemodynamics of blood pressure control during prolonged mental stress. Psychophysiology 2002;39:585-90.
34. Willemsen G, Ring C, Carroll D, Evans P, Clow A, Hucklebridge F. Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic and cold pressor. Psychophysiology 1998;35:252-9.

35. Emons WH, Meijer RR, Denollet J. Negative affectivity and social inhibition in cardiovascular disease: evaluating type-D personality and its assessment using item response theory. J Psychosom Res 2007;63:27-39.

36. Marmot MG, Smith GD, Stansfeld S, Patel C, North F, Head J, White I, Brunner E, Feeney A. Health inequalities among British civil servants: the Whitehall II study. Lancet 1991;337:1387-93.

37. Jurca R, Jackson AS, LaMonte MJ, Morrow JR, Jr., Blair SN, Wareham NJ, Haskell WL, van Mechelen W, Church TS, Jakicic JM, Laukkanen R. Assessing cardiorespiratory fitness without performing exercise testing. Am J Prev Med 2005;29:185-93.

38. Zigmond AS, Snaith RP. The Hospital Anxiety and Depression Scale. Acta Psychiatr Scand 1983;67:361-70.

39. Herrmann C. International experiences with the hospital anxiety and depression scale - A review of validation data and clinical results. J Psychosom Res 1997;42:17-41.

40. Moorey S, Greer S, Watson M, Gorman C, Rowden L, Tunmore R, Robertson B, Bliss J. The factor structure and factor stability of the Hospital Anxiety and Depression Scale in patients with cancer. British Journal of Psychiatry 1991;158:255-9.

41. Heaney JL, Ginty AT, Carroll D, Phillips AC. Preliminary evidence that exercise dependence is associated with blunted cardiac and cortisol reactions to acute psychological stress. Int J Psychophysiol 2011;79:323-9.

42. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol Bull 2004;130:355-91.

43. Grynberg D, Gidron Y, Denollet J, Luminet O. Evidence for a cognitive bias of interpretation toward threat in individuals with a Type D personality. J Behav Med 2012;35:95-102.

44. Kret ME, Denollet J, Grezes J, de Gelder B. The role of negative affectivity and social inhibition in perceiving social threat: an fMRI study. Neuropsychologia 2011;49:1187-93.

45. Bibbey A, Carroll D, Roseboom TJ, Phillips AC, de Rooij SR. Personality and physiological reactions to acute psychological stress. Int J Psychophysiol 2013;90:28-36.

46. Sher L. Type D personality: the heart, stress, and cortisol. QJM 2005;98:323-9.

47. Kamarck TW, Schwartz JE, Shiffman S, Muldoon MF, Sutton-Tyrrell K, Janicki DL. Psychosocial stress and cardiovascular risk: what is the role of daily experience? J Pers 2005;73:1749-74.

48. Marin TJ, Miller GE. The interpersonally sensitive disposition and health: an integrative review. Psychol Bull 2013;139:941-84.

49. Ferguson E, Williams L, O'Connor RC, Howard S, Hughes BM, Johnston DW, Allan JL, O'Connor DB, Lewis CA, Grealy MA, O'Carroll RE. A taxometric analysis of type-D personality. Psychosom Med 2009;71:981-6.

50. Smith TW. Toward a more systematic, cumulative, and applicable science of personality and health: lessons from type D personality. Psychosom Med 2011;73:528-32.

51. Denollet J, Pedersen SS, Vrints CJ, Conraads VM. Predictive value of social inhibition and negative affectivity for cardiovascular events and mortality in patients with coronary artery disease: the type D personality construct. Psychosom Med 2013;75:873-81.

52. Cohen J, Cohen P. Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences. London: Lawrence Erlbaum Associates; 1983.

53. Kupper N, Denollet J. Type D personality as a prognostic factor in heart disease: assessment and mediating mechanisms. J Pers Assess 2007;89:265-76.

54. de Voogd JN, Sanderman R, Coyne JC. A meta-analysis of spurious associations between type D personality and cardiovascular disease endpoints. Ann Behav Med 2012;44:136-7.

55. Pruessner JC, Gaab J, Hellhammer DH, Lintz D, Schommer N, Kirschbaum C. Increasing correlations between personality traits and cortisol stress responses obtained by data aggregation. Psychoneuroendocrinology 1997;22:615-25.

TABLE 1. Characteristics and Stress Task Ratings and Performance of Type D and Non-Type D Participants Stratified by Non-social and Social conditions.

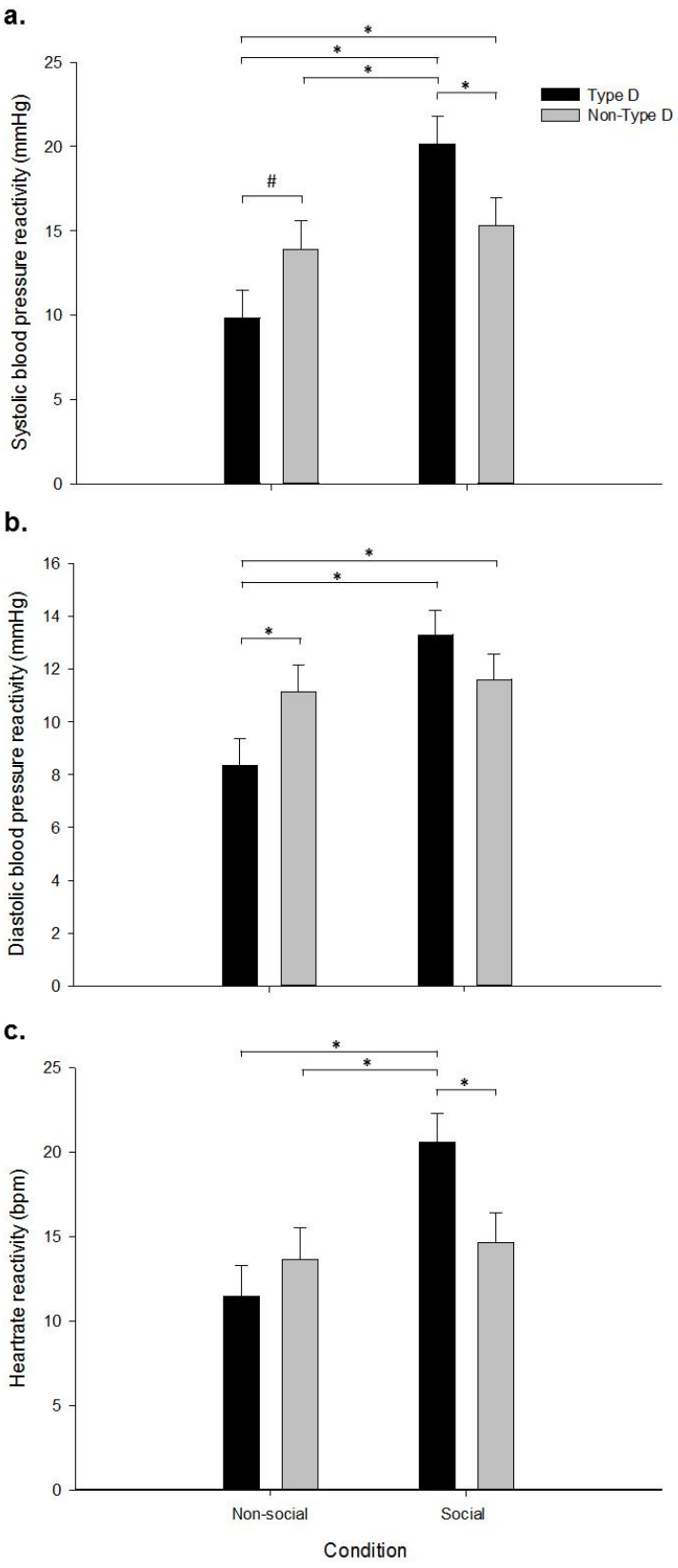
	Non-social		Social	
	Type D	Non-Type D	Type D	Non-Type D
	Mean (SD)/N (%)			
N	31 (24)	30 (23)	35 (27)	34 (26)
DS14 total score	38.4 (4.88)	7.1 (2.83)	39.9 (5.50)	6.4 (2.79)
Gender (females)	18 (58)	14 (47)	18 (51)	20 (59)
Age (years)	21.5 (2.41)	19.9 (1.53)	20.4 (1.91)	20.1 (1.17)
BMI (kg/m^2)	22.9 (2.45)	22.8 (3.10)	22.7 (3.57)	22.8 (2.62)
HADS (depression subscale)	5.6 (3.11)	1.6 (1.63)	6.2 (3.96)	1.7 (1.96)
Current smoker	6 (19)	2 (7)	5 (14)	7 (21)
Units of alcohol per week (≥11)	2 (7)	9 (30)	10 (29)	9 (27)
Cardio-respiratory fitness (METS)	12.7 (2.06)	14.2 (1.95)	12.9 (1.96)	13.8 (2.18)
Stressfulness	4.2 (1.32)	3.5 (1.23)	4.4 (1.07)	3.9 (1.31)
Engagement	4.2 (1.53)	4.5 (1.31)	4.4 (1.21)	4.3 (1.42)
Number of Stroop trials	104.7 (13.81)	109.2 (12.61)	107.6 (13.00)	106.1 (10.79)
Correct responses to Stroop (%)	58.0 (7.31)	58.3 (5.99)	57.2 (6.12)	56.6 (6.09)
PASAT total score	672.6 (147.17)	685.7 (148.00)	728.1 (139.55)	677.5 (117.69)

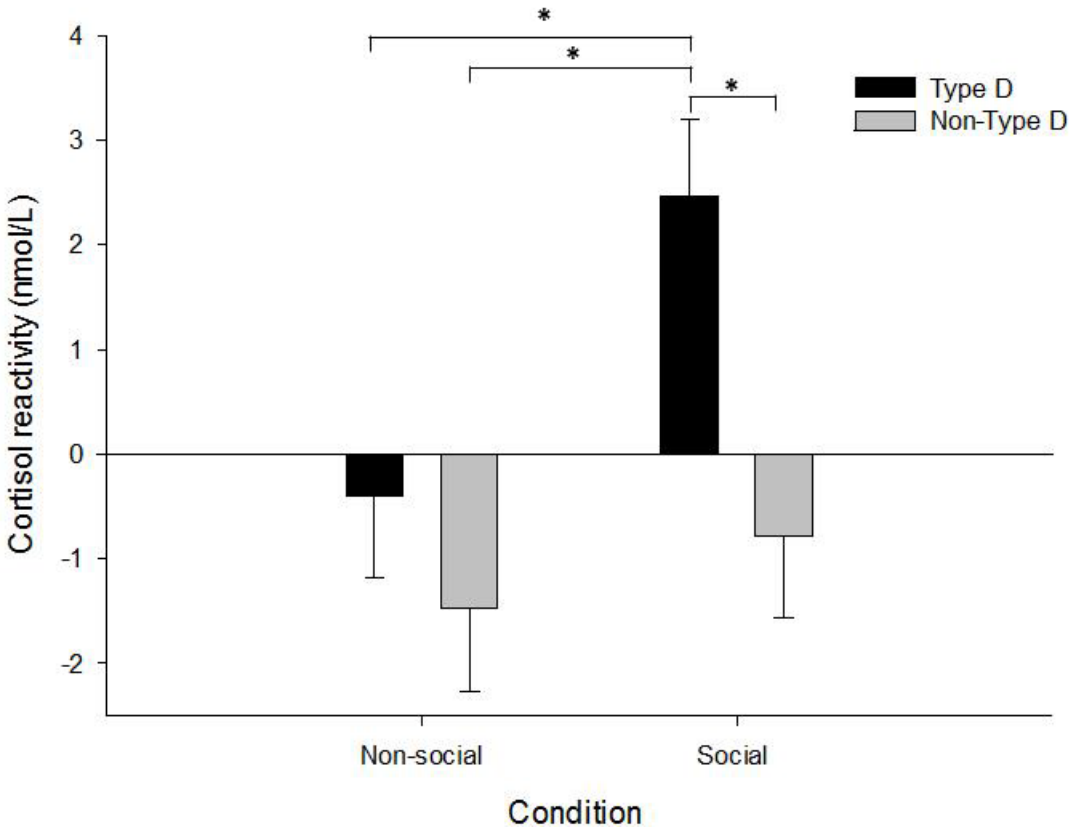
TABLE 2. Baseline and Stress Levels for Cardiovascular and Cortisol Parameters of Type D and Non-Type D Participants Stratified by Non-social and Social conditions.

	Non-social		Social	
	Type D	Non-Type D	Type D	Non-Type D
	Mean (SD)			
SBP baseline (mmHg)	103.3 (10.79)	103.1 (9.13)	104.8 (10.18)	104.2 (8.00)
DBP baseline (mmHg)	52.4 (4.95)	52.4 (4.90)	55.0 (6.44)	53.9 (5.23)
HR baseline (mmHg)	64.1 (9.51)	61.9 (9.20)	65.8 (10.45)	60.5 (11.41)
Cortisol baseline (nmol/L)	7.2 (4.56)	7.8 (5.22)	8.5 (3.42)	10.0 (6.60)
SBP stress (mmHg)	113.2 (13.31)	117.0 (11.19)	125.0 (18.18)	119.5 (14.48)
DBP stress (mmHg)	62.4 (7.06)	63.5 (6.03)	68.3 (8.04)	65.5 (7.48)
HR stress (mmHg)	75.5 (12.09)	75.5 (10.23)	86.4 (17.72)	75.2 (14.08)
Cortisol stress (nmol/L)	6.8 (4.29)	6.3 (4.30)	11.0 (7.20)	9.2 (5.57)

Figure 1. Mean (SE) (a) systolic blood pressure, (b) diastolic blood pressure, and (c) heart rate reactivity levels across social manipulation and Type D classification. * $p \leq .05$, # $p \leq .10$.

Figure 2. Mean (SE) salivary cortisol reactivity levels across social manipulation and Type D classification. * $p \leq .05$.


a.
Systolic blood pressure reactivity (mmHg)
Type D
Non-Type D
b.
Diastolic blood pressure reactivity (mmHg)
c.
Heartrate reactivity (bpm)
Non-social
Social
Condition


*
*
*
Type D
Non-Type D
Cortisol reactivity (nmol/L)
4
3
2
1
0
-1
-2
Non-social
Social
Condition